CLAIMS

1.   A solid support, comprising:

         (A) at least one lymphocyte affecting molecule; and

         (B) at least one molecular complex that, when bound to an antigen, engages a

unique clonotypic lymphocyte receptor.

2.   The solid support of claim 1 wherein the solid support is a rigid solid support or a flexible

solid support.

3.   The solid support of claim 2 which is a rigid solid support, wherein the rigid solid support

is a particle.

4.   The solid support of claim 1 wherein the at least one lymphocyte affecting molecule is a

T cell affecting molecule and wherein the molecular complex is an antigen presenting complex

that comprises at least one antigen binding cleft.

5.   The solid support of claim 4 wherein the at least one antigen presenting complex

comprises an MHC class I peptide binding cleft.

6.   The solid support of claim 5 wherein the at least one antigen presenting complex is an

MHC class I molecule.

7.   The solid support of claim 5 wherein the at least one antigen presenting complex is an

MHC class I molecular complex comprising at least two fusion proteins, wherein a first fusion

protein comprises a first MHC class I α chain and a first immunoglobulin heavy chain and

wherein a second fusion protein comprises a second MHC class I α chain and a second

immunoglobulin heavy chain, wherein the first and second immunoglobulin heavy chains associate to form the MHC class I molecular complex, wherein the MHC class I molecular complex comprises a first MHC class I peptide binding cleft and a second MHC class I peptide binding cleft.

8. The solid support of claim 4 wherein the at least one antigen presenting complex comprises an MHC class II peptide binding cleft.

9. The solid support of claim 8 wherein the antigen presenting complex is an MHC class II molecule.

10. The solid support of claim 8 wherein the antigen presenting complex is an MHC class II molecular complex comprising at least four fusion proteins, wherein:

> (a) two first fusion proteins comprise (i) an immunoglobulin heavy chain and (ii) an extracellular domain of an MHC class IIβ chain; and

> (b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) an extracellular domain of an MHC class IIα chain,

wherein the two first and the two second fusion proteins associate to form the MHC class II molecular complex, wherein the extracellular domain of the MHC class IIβ chain of each first fusion protein and the extracellular domain of the MHC class IIα chain of each second fusion protein form an MHC class II peptide binding cleft.

11. The solid support of claim 10 wherein the immunoglobulin heavy chain comprises a variable region.

12. The solid support of claim 4 wherein an antigenic peptide is bound to the at least one antigen binding cleft.

13. The solid support of claim 12 wherein the antigenic peptide is selected from the group consisting of a peptide of a tumor-associated antigen, a peptide of an autoantigen, a peptide of an alloantigen, and a peptide of an infectious agent antigen.

14. The solid support of claim 4 comprising at least two antigen presenting complexes.

15. The solid support of claim 14 wherein an identical antigen is bound to each antigen binding cleft of the at least two antigen presenting complexes.

16. The solid support of claim 14 wherein different antigens are bound to each antigen binding cleft of the at least two antigen presenting complexes.

17. The solid support of claim 14 wherein a first antigen presenting complex comprises at least one MHC class I peptide binding cleft and wherein a second antigen presenting complex comprises at least one MHC class II peptide binding cleft.

18. The solid support of claim 17 wherein identical antigenic peptides are bound to the at least one MHC class I peptide binding cleft and the at least one MHC class II peptide binding cleft.

19. The solid support of claim 17 wherein different antigenic peptides are bound to the at least one MHC class I peptide binding cleft and the at least one MHC class II peptide binding cleft.

20. The solid support of claim 4 wherein the at least one antigen presenting complex is a non-classical MHC-like molecule.

21. The solid support of claim 20 wherein the non-classical MHC-like molecule is a CD1 family member.

22. The solid support of claim 21 wherein the non-classical MHC-like molecule is selected from the group consisting of CD1a, CD1b, CD1c, CD1d, and CD1e.

23. The solid support of claim 4 wherein the at least one T cell affecting molecule is a T cell costimulatory molecule.

24. The solid support of claim 23 wherein the T cell costimulatory molecule is selected from the group consisting of CD80 (B7-1), CD86 (B7-2), B7-H3, 4-1BBL, CD27, CD30, CD134 (OX-40L), B7h (B7RP-1), CD40, LIGHT, an antibody that specifically binds to CD28, an antibody that specifically binds to HVEM, an antibody that specifically binds to CD40L, an antibody that specifically binds to OX40, and an antibody that specifically binds to 4-1BB.

25. The solid support of claim 4 wherein the at least one T cell affecting molecule is an adhesion molecule.

26. The solid support of claim 25 wherein the adhesion molecule is selected from the group consisting of ICAM-1 and LFA-3.

27. The solid support of claim 4 wherein the at least one T cell affecting molecule is a T cell growth factor.

28. The solid support of claim 27 wherein the T cell growth factor is selected from the group consisting of a cytokine and a superantigen.

29. The solid support of claim 28 wherein the T cell growth factor is a cytokine and the cytokine is selected from the group consisting of IL-2, IL-4, IL-7, IL-10, IL-12, IL-15, and gamma interferon.

30. The solid support of claim 27 wherein the T cell growth factor is selected from the group consisting of:

(A) a first molecular complex comprising at least two fusion proteins, wherein a first fusion protein comprises a first cytokine and an immunoglobulin heavy chain and wherein a second fusion protein comprises a second cytokine and a second immunoglobulin heavy chain, wherein the first and second immunoglobulin heavy chains associate to form the first molecular complex; and

(B) a second molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) an immunoglobulin heavy chain and (ii) a first cytokine; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) a second cytokine,

wherein the two first and the two second fusion proteins associate to form the second molecular complex.

31. The solid support of claim 30 wherein the T cell growth factor is the first molecular complex.

32. The solid support of claim 31 wherein the first and second cytokines are identical.

33. The solid support of claim 31 wherein the first and second cytokines are different.

34. The solid support of claim 30 wherein the T cell growth factor is the second molecular complex.

35. The solid support of claim 34 wherein the first and second cytokines are identical.

36. The solid support of claim 34 wherein the first and second cytokines are different.

37. The solid support of claim 4 wherein the at least one T cell affecting molecule is a regulatory T cell inducer molecule.

38. The solid support of claim 37 wherein the at least one regulatory T cell inducer molecule is selected from the group consisting of TGFβ, IL-10, interferon-α, and IL-15.

39. The solid support of claim 4 wherein the at least one T cell affecting molecule is an apoptosis-inducing molecule.

40. The solid support of claim 39 wherein the apoptosis-inducing molecule is selected from the group consisting of a toxin, TNFα, and Fas ligand.

41. The solid support of claim 4 which comprises at least two different T cell affecting molecules.

42. A solid support comprising:

(A) at least one B cell affecting molecule; and

(B) at least one molecular complex that engages B cell surface immunoglobulins or MHC-antigen complexes on a B cell surface.

43.   The solid support of claim 42 wherein the at least one B cell affecting molecule is CD40 ligand.

44.   The solid support of claim 42 wherein the molecular complex is a T cell receptor (TCR).

45.   The solid support of claim 42 wherein the molecular complex is a TCR molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) a TCR α chain or a TCR γ chain; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) an extracellular domain of a TCR β chain or a TCR δ chain, wherein if the two first fusion proteins comprise the TCR α chain, then the two second fusion proteins comprising the TCR β chain and wherein if the two first fusion proteins comprise the TCR γ chain, then the two second fusion proteins comprising the TCR δ chain,

wherein the two first and the two second fusion proteins associate to form the TCR molecular complex, wherein the extracellular domain of the TCR α or γ chain of each first fusion protein and the extracellular domain of the TCR β or δ chain of each second fusion protein form a TCR antigen binding cleft.

46.   A particle, comprising:

(A) at least one T cell costimulatory molecule; and

(B) at least one MHC class I molecular complex comprising at least two fusion proteins, wherein a first fusion protein comprises a first MHC class I α chain and a first immunoglobulin heavy chain and wherein a second fusion protein comprises a second MHC class I α chain and a second immunoglobulin heavy chain, wherein the first and second immunoglobulin heavy chains associate to form the MHC class I molecular complex, wherein the MHC class I molecular complex comprises a first MHC class I peptide binding cleft and a second MHC class I peptide binding cleft.

47.   The particle of claim 46 wherein the at least one T cell costimulatory molecule is an antibody that specifically binds to CD28.

48.   A preparation comprising a plurality of particles, wherein particles of the plurality comprise:

(A) at least one lymphocyte affecting molecule; and

(B) at least one molecular complex that, when bound to an antigen, engages a unique clonotypic lymphocyte receptor.

49.   The preparation of claim 48 further comprising a pharmaceutically acceptable carrier.

50.   The preparation of claim 48 wherein the at least one lymphocyte affecting molecule is a T cell affecting molecule and the at least one molecular complex is an antigen presenting complex that comprises at least one antigen binding cleft.

51.   The preparation of claim 48 wherein the plurality of particles comprises:

(A) at least one first particle wherein the at least one antigen binding cleft of the first particle is an MHC class I peptide binding cleft; and

(B) at least one second particle wherein the at least one antigen binding cleft is an MHC class II peptide binding cleft.

52. The preparation of claim 51 wherein an antigenic peptide is bound to the at least one peptide binding cleft of the first particle.

53. The preparation of claim 51 wherein a first antigenic peptide is bound to the at least one peptide binding cleft of the first particle and a second antigenic peptide is bound to the at least one peptide binding cleft of the second particle.

54. The preparation of claim 53 wherein the first and second antigenic peptides are identical.

55. The preparation of claim 53 wherein the first and second antigenic peptides are different.

56. The preparation of claim 50 wherein each antigen binding cleft of the antigen presenting complexes is an MHC class I peptide binding cleft.

57. The preparation of claim 56 wherein antigenic peptides are bound to the MHC class I peptide binding clefts.

58. The preparation of claim 57 wherein the antigenic peptides are identical.

59. The preparation of claim 57 wherein the antigenic peptides are different.

60. The preparation of claim 50 wherein each antigen binding cleft of the antigen presenting complexes is an MHC class II peptide binding cleft.

61.   The preparation of claim 60 wherein antigenic peptides are bound to the MHC class II peptide binding clefts.

62.   The preparation of claim 61 wherein the antigenic peptides are identical.

63.   The preparation of claim 61 wherein the antigenic peptides are different.

64.   The preparation of claim 50 wherein the antigen presenting complex is an MHC class I molecular complex comprising at least two fusion proteins, wherein a first fusion protein comprises a first MHC class I α chain and a first immunoglobulin heavy chain and wherein a second fusion protein comprises a second MHC class I α chain and a second immunoglobulin heavy chain, wherein the first and second immunoglobulin heavy chains associate to form the MHC class I molecular complex, wherein the MHC class I molecular complex comprises a first MHC class I peptide binding cleft and a second MHC class I peptide binding cleft.

65.   The preparation of claim 50 wherein the antigen presenting complex is an MHC class II molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) an immunoglobulin heavy chain and (ii) an extracellular domain of an MHC class IIβ chain; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) an extracellular domain of an MHC class IIα chain,

wherein the two first and the two second fusion proteins associate to form the MHC class II molecular complex, wherein the extracellular domain of the MHC class IIβ chain of each first

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fusion protein and the extracellular domain of the MHC class IIα chain of each second fusion protein form an MHC class II peptide binding cleft.

66. A preparation comprising a plurality of particles, wherein particles of the plurality comprise:

(A) at least one B cell affecting molecule; and

(B) at least one molecular complex that engages B cell surface immunoglobulins or MHC-antigen complexes on a B cell surface.

67. The preparation of claim 66 wherein the molecular complex is a TCR molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) a TCR α chain or a TCR γ chain; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) an extracellular domain of a TCR β chain or a TCR δ chain, wherein if the two first fusion proteins comprise the TCR α chain, then the two second fusion proteins comprising the TCR β chain and wherein if the two first fusion proteins comprise the TCR γ chain, then the two second fusion proteins comprising the TCR δ chain,

wherein the two first and the two second fusion proteins associate to form the TCR molecular complex, wherein the extracellular domain of the TCR α or γ chain of each first fusion protein and the extracellular domain of the TCR β or δ chain of each second fusion protein form a TCR antigen binding cleft.

68. The particle of claim 3 which is magnetic.

69.   The particle of claim 3 which is biodegradable.

70.   The particle of claim 3 which is plastic.

71.   A method of inducing the formation of antigen-specific T cells, comprising the step of: contacting an isolated preparation comprising a plurality of precursor T cells with at least one first solid support of claim 4, wherein antigens are bound to the antigenic binding clefts, thereby inducing members of the plurality of precursor T cells to form a first cell population comprising antigen-specific T cells that recognize the antigen, wherein the number or percentage of antigen-specific T cells in the first cell population is greater than the number or percentage of antigen-specific T cells that are formed if precursor T cells are incubated with a solid support that comprises an antibody that specifically binds to CD3 but does not comprise an antigen presenting complex.

72.   The method of claim 71 wherein the antigen-specific T cells are cytotoxic T cells.

73.   The method of claim 71 wherein the antigen-specific T cells are helper T cells.

74.   The method of claim 71 wherein the antigen-specific T cells are regulatory T cells.

75.   The method of claim 71 further comprising the step of separating the antigen-specific T cells from the first cell population.

76.   The method of claim 71 further comprising the step of incubating the first cell population with at least one second solid support of claim 4, wherein antigens are bound to the antigen binding clefts of the particles, wherein the step of incubating is carried out for a period of time sufficient to form a second cell population comprising an increased number or percentage of antigen-specific T cells relative to the number or percentage of antigen-specific T cells in the first cell population.

77.   The method of claim 71 wherein the antigens are identical.

78.   The method of claim 71 wherein the antigens are different.

79.   The method of claim 71 wherein the isolated preparation is contacted with at least two first solid supports, wherein different antigens are bound to each of the first solid supports.

80.   A method of increasing the number or percentage of antigen-specific T cells in a population of cells, comprising the step of:

incubating a first cell population comprising antigen-specific T cells with at least one first solid support of claim 4, wherein antigens are bound to the antigen binding clefts, wherein the step of incubating is carried out for a period of time sufficient to form a second cell population comprising an increased number or percentage of antigen-specific T cells relative to the number or percentage of antigen-specific T cells in the first cell population.

81.   The method of claim 80 wherein the first cell population is a homogeneous cell population.

82.   The method of claim 71 further comprising the step of administering the antigen-specific T cells to a patient.

83.   The method of claim 82 wherein the patient has cancer, an autoimmune disease, an infectious disease, or is immunosuppressed.

84.   The method of claim 82 wherein the precursor T cells are obtained from the patient.

85.   The method of claim 82 wherein the precursor T cells are obtained from a donor who is not the patient.

86.   The method of claim 82 wherein the antigen-specific T cells are administered by a route of administration selected from the group consisting of intravenous administration, intra-arterial

administration, subcutaneous administration, intradermal administration, intralymphatic administration, and intra-tumoral administration.

87.   The method of claim 80 further comprising the step of administering the antigen-specific T cells of the second population to the patient.

88.   A method of regulating an immune response in a patient, comprising the step of:

administering to a patient a preparation comprising (A) a plurality of particles and (B) a pharmaceutically acceptable carrier, wherein members of the plurality of particles comprise:

(1) at least one T cell affecting molecule; and

(2) at least one antigen presenting complex, wherein the at least one antigen presenting complex comprises at least one antigen binding cleft, wherein an antigen is bound to the at least one antigen binding cleft.

89.   The method of claim 88 wherein the at least one T cell affecting molecule is selected from the group consisting of (1) an apoptosis-inducing molecule, (2) a regulatory T cell inducing molecule, (3) a T cell costimulatory molecule, (4) an adhesion molecule, and (5) a T cell growth factor.

90.   A method of suppressing an immune response in a patient, comprising the steps of:

administering to a patient a preparation comprising (A) a plurality of particles and (B) a pharmaceutically acceptable carrier, wherein members of the plurality of particles comprise:

(1) at least one apoptosis-inducing molecule; and

(2) at least one antigen presenting complex, wherein the at least one antigen presenting complex comprises at least one antigen binding cleft, wherein an antigen is bound to the at least one antigen binding cleft.

91.   A cell, comprising:

(A) at least one lymphocyte affecting molecule; and

(B) at least one molecular complex that, when bound to an antigen, engages a unique clonotypic lymphocyte receptor.

92.   The cell of claim 91 wherein the at least one lymphocyte affecting molecule is a T cell affecting molecule and wherein the at least one molecular complex is an antigen presenting complex comprising at least one peptide binding cleft, wherein the antigen presenting complex is selected from the group consisting of:

(1) an MHC class I molecular complex comprising at least two fusion proteins, wherein a first fusion protein comprises a first MHC class I α chain and a first immunoglobulin heavy chain and wherein a second fusion protein comprises a second MHC class I α chain and a second immunoglobulin heavy chain, wherein the first and second immunoglobulin heavy chains associate to form the MHC class I molecular complex, wherein the MHC class I molecular complex comprises a first MHC class I peptide binding cleft and a second MHC class I peptide binding cleft; and

(2) an MHC class II molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) an immunoglobulin heavy chain and (ii) an extracellular domain of an MHC class IIβ chain; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) an extracellular domain of an MHC class IIα chain,

wherein the two first and the two second fusion proteins associate to form the MHC class II molecular complex, wherein the extracellular domain of the MHC class IIβ chain of each first fusion protein and the extracellular domain of the MHC class IIα chain of each second fusion protein form an MHC class II peptide binding cleft.

93.   The cell of claim 92 wherein the antigen presenting complex is an MHC class II molecular complex and wherein the immunoglobulin heavy chain comprises a variable region.

94.   The cell of claim 92 wherein an antigenic peptide is bound to the at least one peptide binding cleft.

95.   The cell of claim 94 wherein the antigenic peptide is selected from the group consisting of a peptide of a tumor-associated antigen, a peptide of an autoantigen, a peptide of an alloantigen, and a peptide of an infectious agent antigen.

96.   The cell of claim 92 comprising at least two antigen presenting complexes.

97.   The cell of claim 96 wherein identical antigenic peptides are bound to each peptide binding cleft of the at least two antigen presenting complexes.

98.   The cell of claim 96 wherein different antigenic peptides are bound to each peptide binding cleft of the at least two antigen presenting complexes.

99.   The cell of claim 96 wherein a first antigen presenting complex is an MHC class I molecular complex and wherein a second antigen presenting complex is an MHC class II molecular complex.

100.   The cell of claim 99 wherein identical antigenic peptides are bound to the peptide binding clefts of the at least one MHC class I molecular complex and the peptide binding clefts of the at least one MHC class II molecular complex.

101.   The cell of claim 99 wherein different antigenic peptides are bound to the peptide binding clefts of the at least one MHC class I molecular complex and the peptide binding clefts of the at least one MHC class II molecular complex.

102.   The cell of claim 92 wherein the at least one T cell affecting molecule is a T cell costimulatory molecule.

103.   The cell of claim 102 wherein the T cell costimulatory molecule is selected from the group consisting of CD80 (B7-1), CD86 (B7-2), B7-H3, 4-1BBL, CD27, CD30, CD134 (OX-40L), B7h (B7RP-1), CD40, LIGHT, an antibody that specifically binds to CD28, an antibody that specifically binds to HVEM, an antibody that specifically binds to CD40L, an antibody that specifically binds to OX40, and an antibody that specifically binds to 4-1BB.

104.   The cell of claim 92 wherein the at least one T cell affecting molecule is an adhesion molecule.

105.   The cell of claim 104 wherein the adhesion molecule is selected from the group consisting of ICAM-1, LFA-3, and LFA-1.

106. The cell of claim 92 wherein the at least one T cell affecting molecule is a T cell growth factor.

107. The cell of claim 106 wherein the T cell growth factor is selected from the group consisting of a cytokine and a superantigen.

108. The cell of claim 106 wherein the T cell growth factor is a cytokine and the cytokine is selected from the group consisting of IL-2, IL-4, IL-7, IL-10, IL-12, IL-15, and gamma interferon.

109. The cell of claim 106 wherein the at least one T cell affecting molecule is selected from the group consisting of:

(A) a first molecular complex comprising at least two fusion proteins, wherein a first fusion protein comprises a first cytokine and an immunoglobulin heavy chain and wherein a second fusion protein comprises a second cytokine and a second immunoglobulin heavy chain, wherein the first and second immunoglobulin heavy chains associate to form the first molecular complex; and

(B) a second molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) an immunoglobulin heavy chain and (ii) a first cytokine; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) a second cytokine,

wherein the two first and the two second fusion proteins associate to form the second molecular complex.

110. The cell of claim 109 wherein the first and second cytokines are identical.

111. The cell of claim 109 wherein the first and second cytokines are different.

112. The cell of claim 92 wherein the at least one exogenous T cell affecting molecule is a regulatory T cell inducer molecule.

113. The cell of claim 112 wherein the at least one regulatory T cell inducer molecule is selected from the group consisting of TGFβ, IL-10, interferon-α, and IL-15.

114. The cell of claim 92 which comprises at least two different T cell affecting molecules.

115. The cell of claim 91 wherein the at least one lymphocyte affecting molecule is a B cell affecting molecule and wherein the at least one molecular complex is a TCR molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) a TCR α chain or a TCR γ chain; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) an extracellular domain of a TCR β chain or a TCR δ chain, wherein if the two first fusion proteins comprise the TCR α chain, then the two second fusion proteins comprising the TCR β chain and wherein if the two first fusion proteins comprise the TCR γ chain, then the two second fusion proteins comprising the TCR δ chain,

wherein the two first and the two second fusion proteins associate to form the TCR molecular complex, wherein the extracellular domain of the TCR α or γ chain of each first fusion protein

and the extracellular domain of the TCR β or δ chain of each second fusion protein form a TCR antigen binding cleft.

116.   A preparation comprising a plurality of the cells of claim 91.

117.   The preparation of claim 116 further comprising a pharmaceutically acceptable carrier.

118.   A method of inducing the formation of antigen-specific T cells, comprising the step of:

contacting an isolated preparation comprising a plurality of precursor T cells with a first plurality of the cells of claim 92, wherein antigenic peptides are bound to the peptide binding clefts, thereby inducing members of the plurality of precursor T cells to form a first cell population comprising antigen-specific T cells that recognize the antigenic peptide, wherein the number or percentage of antigen-specific T cells in the first cell population is greater than the number or percentage of antigen-specific T cells that are formed if precursor T cells are incubated with a second plurality of cells, wherein the cells of the second plurality comprise an antibody that specifically binds to CD3 but do not comprise an antigen presenting complex.

119.   The method of claim 118 wherein the antigen-specific T cells are cytotoxic T cells.

120.   The method of claim 118 wherein the antigen-specific T cells are helper T cells.

121.   The method of claim 118 wherein the antigen-specific T cells are regulatory T cells.

122.   The method of claim 118 further comprising the step of separating the antigen-specific T cells from the first cell population.

123.   The method of claim 118 further comprising the step of incubating the first cell population with a second plurality of the cells of claim 1, wherein antigenic peptides are bound

to the peptide binding clefts of the cells, wherein the step of incubating is carried out for a period of time sufficient to form a second cell population comprising an increased number or percentage of antigen-specific T cells relative to the number or percentage of antigen-specific T cells in the first cell population.

124. A method of increasing the number or percentage of antigen-specific T cells in a population of cells, comprising the step of:

incubating a first cell population comprising antigen-specific T cells with a plurality of the cells of claim 92, wherein antigenic peptides are bound to the peptide binding clefts of the cells, wherein the step of incubating is carried out for a period of time sufficient to form a second cell population comprising an increased number or percentage of antigen-specific T cells relative to the number or percentage of antigen-specific T cells in the first cell population.

125. The method of claim 124 wherein the first cell population is a homogeneous cell population.

126. The method of claim 118 further comprising the step of administering the antigen-specific T cells to a patient.

127. The method of claim 126 wherein the patient has cancer, an autoimmune disease, an infectious disease, or is immunosuppressed.

128. The method of claim 126 wherein the precursor T cells are obtained from the patient.

129. The method of claim 126 wherein the precursor T cells are obtained from a donor who is not the patient.

130. The method of claim 126 wherein the antigen-specific T cells are administered by a route of administration selected from the group consisting of intravenous administration, intra-arterial

administration, subcutaneous administration, intradermal administration, intralymphatic administration, and intra-tumoral administration.

131. The method of claim 124 further comprising the step of administering the antigen-specific T cells of the second population to the patient.

132. A method of regulating an immune response in a patient, comprising the step of:

administering to a patient a preparation comprising a plurality of the cells of claim 92 and a pharmaceutically acceptable carrier, wherein an antigenic peptide is bound to the at least one peptide binding cleft.

133. The method of claim 132 wherein the at least one T cell affecting molecule is selected from the group consisting of (1) an apoptosis-inducing molecule, (2) a regulatory T cell inducing molecule, (3) a T cell costimulatory molecule, (4) an adhesion molecule, and (5) a T cell growth factor.

134. A method of increasing the number or percentage of antibody-producing B cells in a population, comprising the steps of:

contacting an isolated preparation comprising a plurality of precursor B cells with at least one first solid support of claim 42, thereby inducing members of the plurality of precursor B cells to form a first cell population comprising antibody-producing B cells that produce antibodies that specifically bind to the antigenic peptide.

135. The method of claim 134 further comprising the step of separating the B cells that produce the antibodies from the first cell population.

136. The method of claim 134 further comprising the step of incubating the first cell population with at least one second solid support of claim 42, wherein the step of incubating is

carried out for a period of time sufficient to form a second cell population comprising an increased number or percentage of antibody-producing B cells relative to the number or percentage of antibody-producing B cells in the first cell population.

137.   A method of increasing the number or percentage of antibody-producing B cells in a population, comprising the step of:

incubating a first cell population comprising antibody-producing B cells with at least one first solid support of claim 42, wherein the step of incubating is carried out for a period of time sufficient to form a second cell population comprising an increased number or percentage of antibody-producing B cells relative to the number or percentage of antibody-producing B cells in the first cell population.

138.   The method of claim 137 wherein the first cell population is a homogeneous cell population.

139.   A method of increasing the number or percentage of antibody-producing B cells in a population, comprising the steps of:

contacting an isolated preparation comprising a plurality of precursor B cells with the preparation of claim 66, thereby inducing members of the plurality of precursor B cells to form a first cell population comprising antibody-producing B cells that produce antibodies that specifically bind to the antigenic peptide.

140.   A method of regulating an immune response in a patient, comprising the step of:

administering to a patient a preparation comprising (A) a plurality of particles and (B) a pharmaceutically acceptable carrier, wherein members of the plurality of particles comprise:

(1) at least one B cell affecting molecule; and

(2) at least one molecular complex that engages MHC-antigen complexes on a B cell surface.

141.   The method of claim 140 wherein the at least one B cell affecting molecule is selected from the group consisting of (1) CD40 ligand, (2) a cytokine, and (3) a cytokine molecular complex.

142.   The method of claim 140 wherein the molecular complex is selected from the group consisting of a T cell receptor and a TCR molecular complex comprising at least four fusion proteins, wherein:

(a) two first fusion proteins comprise (i) a TCR α chain or a TCR γ chain; and

(b) two second fusion proteins comprise (i) an immunoglobulin light chain and (ii) an extracellular domain of a TCR β chain or a TCR δ chain, wherein if the two first fusion proteins comprise the TCR α chain, then the two second fusion proteins comprising the TCR β chain and wherein if the two first fusion proteins comprise the TCR γ chain, then the two second fusion proteins comprising the TCR δ chain,

wherein the two first and the two second fusion proteins associate to form the TCR molecular complex, wherein the extracellular domain of the TCR α or γ chain of each first fusion protein and the extracellular domain of the TCR β or δ chain of each second fusion protein form a TCR antigen binding cleft.